

DISEASE AND WINTER FEEDING OF ELK AND BISON:

**A REVIEW AND RECOMMENDATIONS PERTINENT TO THE
JACKSON BISON AND ELK MANAGEMENT PLAN AND
ENVIRONMENTAL IMPACT STATEMENT**

**Prepared for
The Greater Yellowstone Coalition**

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INTRODUCTION

I was contracted by the Greater Yellowstone Coalition (GYC) to prepare a report on relationships between winter feeding of elk and bison on the National Elk Refuge (NER) and certain existing and potential diseases of those populations. The focus was on how the winter feeding of elk and bison affects transmission, prevalence, and impacts of brucellosis and potentially chronic wasting disease (CWD) in NER and Grand Teton National Park (GTNP) elk and bison. Secondly, the GYC was interested in how Alternatives 4 and 6 in the Draft Bison and Elk Management Plan and Environmental Impact Statement, or BEMP (USDI 2005), prepared by staff of the NER and GTNP, may affect brucellosis and potentially chronic wasting disease (CWD) in NER and GTNP elk and bison.

I spent the last 22 years of my 28-year career with the U.S. Department of Interior as the senior wildlife biologist at the National Elk Refuge in Jackson, Wyoming. Among my responsibilities, I coordinated the refuge's wildlife and habitat management programs. This included coordinating the winter feeding program for 5,000-11,000 elk and a bison herd that grew from 55 animals to nearly 800 bison during my tenure, monitoring disease and mortality of elk, and conducting various research projects concerning elk population dynamics and ecology. I served on the Greater Yellowstone Interagency Brucellosis Committee (GYIBC), the Jackson Hole Cooperative Elk Studies Group, the Jackson Interagency Bison Committee, and provided data for and technical review of sections of the BEMP.

This report addresses topics of interest outlined by the GYC, and may serve to supplement comments by the GYC on the BEMP. The management alternatives and environmental analyses of the BEMP, and consequent decision documents, pertain strictly to lands of the NER and GTNP and to the Jackson elk and bison herds which seasonally occupy those jurisdictions. To be most useful to the NEPA review process, my comments focus on those jurisdictions and herds when possible. However, there are commonalities between the winter feeding programs and related disease concerns at the NER and 22 feedgrounds where the State of Wyoming feeds about 16,000 elk in winter. In fact 3 of those feedgrounds in the Gros Ventre drainage occur within the distribution of the Jackson elk and bison herds. Because elk interchange between those feedgrounds and the NER and share common spring-summer-fall ranges with elk and other cervids from adjacent herds, some of my comments about disease issues necessarily pertain to the Jackson herd as a whole, and more generally to other elk feedgrounds south of Jackson Hole in western Wyoming.

OVERVIEW OF JACKSON ELK AND BISON HERDS

The Jackson elk and bison herds are dynamic, migratory populations that have contact with adjacent populations of conspecifics (other herds of the same species) and other wildlife species within, and to a lesser degree, beyond the 5,200 km² of the Snake River drainage delineated as the Jackson herd unit (Smith and Robbins 1994, Cain et al. 2001). Herd unit fidelity is quite high among Jackson elk (Boyce 1989, Smith and Anderson 2001). Interchange is less than 10% with adjacent herds, as prescribed by the Wyoming Game and Fish Department to delineate a distinct population of elk (Thorne et

al. 1997). However, more recent investigations showed that 32% of elk captured and radiocollared on elk feedgrounds in the Gros Ventre drainage in 2002 and 2003 spent summer in either the Green River Herd Unit (15%) or east of the Continental Divide in the Wiggins Fork Herd Unit (17%, Figure 1). One of 25 radioed female elk remained in

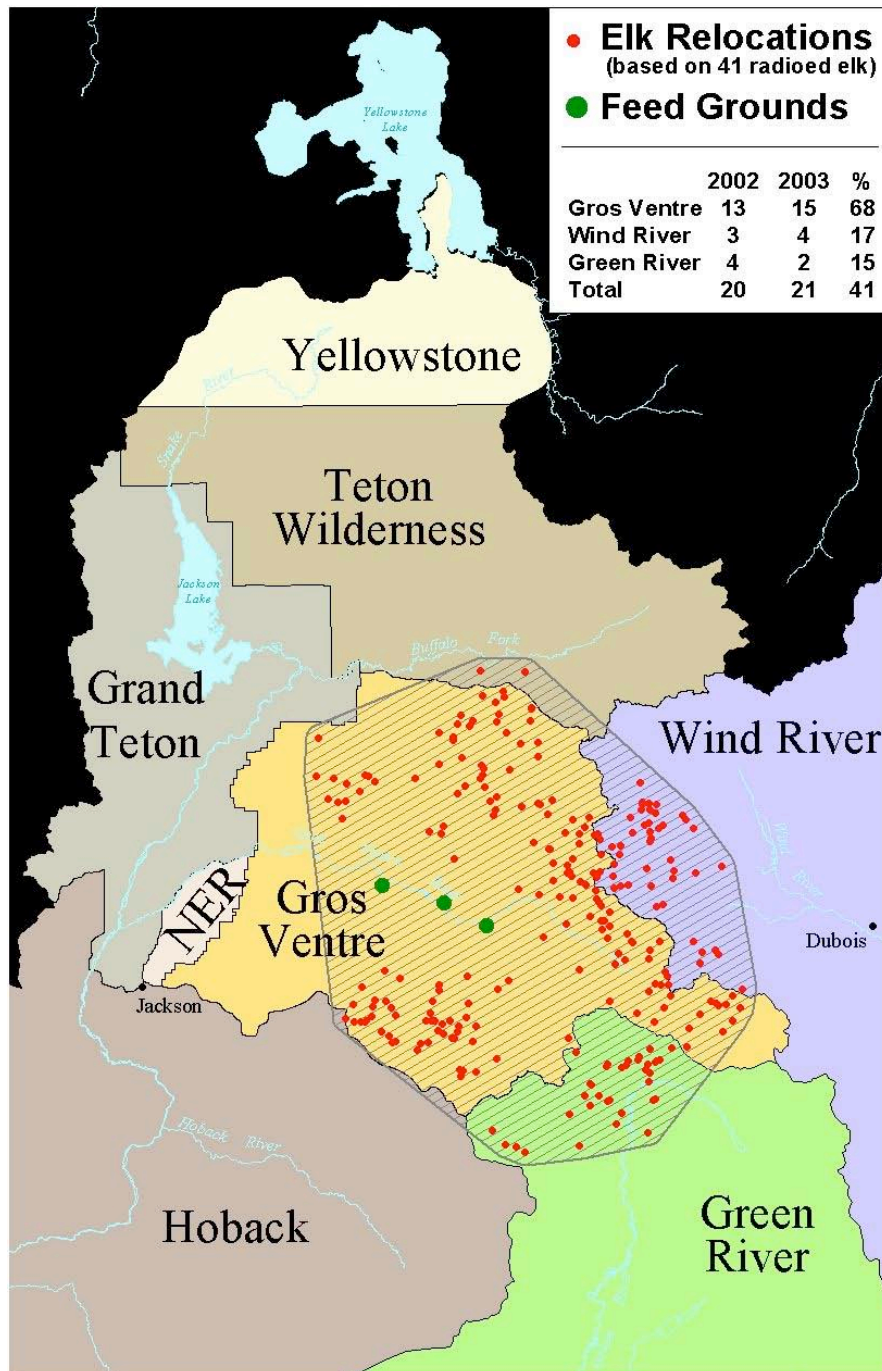


Figure 1. Distribution of summer-fall relocations of radiocollared elk captured on the Alkali and Patrol Cabin elk feedgrounds of the Gros Ventre drainage.

the Wind River drainage yearlong since 2003 (Bruce Smith, unpublished data). Elk from the Jackson herd and elk from east of the Continental Divide (Wiggins Fork and Cody Herds), interchange on shared summer ranges in the eastern Teton Wilderness (Rudd et al. 1983, Queen and Ryder 1996, Smith and Anderson 2001). Conversely, marked elk from Montana's Gravelly-Snowcrest Range have traveled south into Wyoming, including GTNP and the NER (Hamlin and Ross 2002). One such neckbanded animal spent 3 winters on the NER and a radiocollared elk from Montana spent one winter on NER (Bruce Smith, unpublished data). Jackson herd elk also share summer range in Yellowstone National Park with elk from Yellowstone's Northern Range (Craighead et al. 1972) and elk from Idaho's Sand Creek herd (Brown 1985). Thus, an infectious agent endemic in one herd could be transmitted to one or more adjacent herds, or to other susceptible species in or adjacent to the Jackson herds.

Jackson bison also show high fidelity to their seasonal ranges in GTNP and the NER (Cain et al. 2001). However, males have traveled south into the Green River drainage (USDI 2005). In addition, bison from Yellowstone National Park, on at least 2 occasions, joined the Jackson bison herd (Cain et al. 2001).

Evidence of infectious and transmissible diseases in elk and bison of the Greater Yellowstone Area (GYA) have been recognized for many years (Mohler and Eichenhorn 1913, Rush 1932, and Honess and Winter 1956). Much of the discussion of diseases of elk and bison in this report draws on the abundant literature available on the topic. Furthermore, I turned to three recent sources of information that were specifically developed during the BEMP process to address disease issues. These were: 1) an expert panel of administrators, wildlife scientists, and veterinarians convened November 12-14, 2002 to evaluate anticipated disease effects of each of the BEMP's 6 management alternatives; 2) an independent disease impact analysis contracted by the US Fish and Wildlife Service and National Park Service (URS 2003); and 3) an ecological perspective on infectious agents of concern for the Jackson elk and bison populations (Peterson 2003).

As a general model, infectious disease transmission and prevalence are a function of 1) the number and density of infectious animals, 2) the number of susceptible hosts, and 3) conditions which facilitate contact and exposure of susceptible hosts to infectious individuals. The size of the Jackson elk herd ranged from about 9,000 to 18,000 animals during 1982-2002 and provides a mean annual hunter harvest in excess of 3,000 elk (Lubow and Smith 2004). As in most ungulate herds in northern latitudes, densities of animals increase during winter as snow accumulations limit their mobility and forage availability (Demarais and Krausman 2000, Barmore 2003). Densities of elk on the NER are 300-2,500/km², depending on winter severity. Functional density of elk on feedlines reaches 200/ha, exceeding densities on many elk farms (USDI 2005). The introduced Jackson bison herd remained smaller than 25 animals from 1968 to 1975, the year the animals began wintering on the NER. After discovering the elk winter feedgrounds on the NER in 1980, the population underwent a rapid increase to more than 800 animals in 2004. The bison likewise are highly crowded during winter feeding operations (USDI 2005).

Peterson (2003) noted that a "vast array" of parasites could infect Jackson Hole elk and bison and possibly cause disease. Based on suggestions from wildlife biologists

and managers of the Jackson elk and bison populations, he focused his detailed review of infectious agents on those that might be most important to 1) the elk and bison populations, 2) other wildlife populations, and 3) livestock and/or public health in the Jackson Hole area. The URS (2003) report considers a very similar list of diseases. These include documented microparasitic (including brucellosis) and macroparasitic diseases, and undocumented microparasitic diseases (such as bovine tuberculosis and chronic wasting disease).

BRUCELLOSIS

The etiology, pathogenesis, transmission, and biological and economic implications of brucellosis are well understood. For detailed reviews see Thorne (1982), GYIBC (1997), Thorne (2001), and Peterson (2003). In short, elk, bison, and cattle, and a number of other species of mammals are susceptible to infection with the bacterium, *Brucella abortus*, which causes the disease brucellosis.

The hallmark signs of the disease are abortion during the last half of gestation and nonviable calves. Generally, only the first pregnancy after infection results in abortion, and then only in about half of first pregnancies. Oldemeyer et al. (1993) estimated that when 38% of female elk wintering on the NER tested seropositive for brucellosis, calf production would have been diminished by 7% due to brucellosis induced abortions. Elk and bison may also experience synovitis and arthritis which cause lameness in some infected animals. Other clinical signs in bison include retained placentas, orchitis and epididymitis (see Thorne 2001 for details). Horizontal transmission (disease transmission to other herdmates, as opposed to vertical transmission which is from mother to offspring only) may occur when *B. abortus* is ingested from contaminated reproductive products associated with abortions, births, or vaginal discharges.

About 30,000 (3%) of the 1 million free-ranging North American elk are fed by state or federal agencies in winter (Smith 2001). The longest standing winter feeding programs occur in Wyoming where about 75% of these 30,000 elk are fed, and elk are infected with brucellosis at all of the 23 feeding locations that have been tested (19 of 23; GYIBC 1997). Wyoming feedground elk commingle with elk on 2 eastern Idaho feedgrounds that tested positive for brucellosis in 1998 and 1999 (Smith 2001). Elk that share seasonal ranges with feedground elk experience much lower seroprevalences. Where elk are not fed in winter and do not share ranges with infected elk, bison, or cattle, seroprevalence is essentially zero (Thorne 2001, Peterson 2003).

The Jackson elk were first tested and found infected with brucellosis in 1930 (Murie 1951). Elk in Jackson Hole and elsewhere were originally infected with *B. abortus* by infected cattle brought from Europe or by bison that were initially infected by cattle ((Thorne 1982). Peterson et al. (1991) suggest that the source of brucellosis in Jackson bison was either undetected infections in the small herd that was permitted to free-range beginning in 1968 or interspecific transmission of *B. abortus* from elk, most likely in 1980 when bison began sharing feedlines with elk on the NER. Whatever the source, the Jackson elk and bison herds have been chronically infected with brucellosis for decades. The high densities of elk that congregate on the NER and the 3 Gros Ventre feedgrounds perpetuate the disease by exposing large numbers of animals on feedgrounds to *B. abortus* contaminated tissues during the peak period of abortion – February through

May (Thorne 2001, Smith 2001, Peterson 2003). Although all evidence indicates that elk require artificial concentration (e.g. feeding), or periodic re-exposure to infected tissues from elk, bison, or cattle (e.g. Yellowstone National Park elk) to perpetuate the disease (Cheville et al. 1998, Thorne 2001, Peterson 2003), bison are far more efficient at intraspecifically transmitting the disease and can maintain it without fencing or winter feeding (e.g. Yellowstone National Park bison). However, the winter feeding program at the NER probably contributes to the exceptionally high (77 - 84%) seroprevalence among the Jackson bison (GYIBC 1997, USDI 2005). Nonetheless, the Jackson bison herd continues to grow rapidly (USDI 2005).

For a disease that biologically is relatively benign in elk and bison, brucellosis has engendered astonishing costs, controversies, lawsuits, and ill feelings (Smith 2001). The crux of the matter is that brucellosis, which has experimentally been transmitted from elk to cattle and bison to cattle, can cause disease in domestic livestock and/or humans, leads to economic costs to ranchers, and loss of a state's brucellosis class free status which can result in marketing hardships. A national brucellosis program was implemented in 1934 to eradicate brucellosis from cattle and domestic bison herds (Ragan 2002). As that program has neared its stated goal, the focus has shifted to the GYA and its wildlife reservoir of infected elk and bison as a potential source of reinfection of cattle herds (Cheville et al. 1998). Although there is considerable disagreement regarding the risk of *B. abortus* transmission from bison and elk to livestock, regulatory officials surmised that wild elk or bison were the probable source of infection of cattle at six locations in Wyoming prior to 1997 (GYIBC 1997). A brucellosis outbreak in an eastern Idaho cattle herd in 2002 was attributed to infected elk (Ragan 2002). Brucellosis was identified in another eastern Idaho cattle herd in October 2005 and is under investigation (Idaho Department of Agriculture website).

Wyoming lost its brucellosis class free status in February 2004 after a cattle herd in Sublette County (2003) and another in Bighorn County (2004) tested positive for brucellosis (USDI 2005). Two additional cattle herds in Teton County, Wyoming tested positive and were depopulated in 2004 (USDI 2005). The Sublette and Teton County herds occurred within the western Wyoming elk feedground complex, and in the absence of a cattle source for the infections, elk were implicated as the source of the disease. Consequent to legal actions over brucellosis in feedground elk and bison during the 1990s, Smith (2001) noted "Brucellosis has elevated the [wildlife] feeding issue to a new level of public awareness. More citizens question the justification for feeding when the practice is responsible for the spread and maintenance of the disease in elk." Thus, disease management, and management of brucellosis in bison and elk in particular, is one of the four management goals of the BEMP.

REDUCING BRUCELLOSIS AMONG ELK AND BISON

This discussion begins by acknowledging previous admonitions about the vast difference between elimination of *B. abortus* from feedground elk and bison and reducing prevalence in herds to some low level (Keiter and Froelicher 1993, Cheville et al. 1998). Secondly, I treat elk and bison separately, given the differences in social behavior and consequent differences in transmission and maintenance of infections in the two species.

Elk

Keiter and Froelicher (1993) reviewed the lawsuit brought against the federal government by the Parker Land and Cattle Company after Parker's cattle herd became infected with brucellosis, allegedly as a result of contact with brucellosis infected wild bison and/or elk. They suggested the only fully effective means of eradicating brucellosis from the GYA's elk and bison would be depopulation, "an extreme policy choice, with serious political, ecological, and economic repercussions." Peterson (2003) and others agree with their perspective on eradication.

In 1994 the GYIBC formulated and adopted a position statement that recognizes the link between concentration of ungulates at feedgrounds and disease problems. The statement concludes, "...the GYIBC strongly recommends that winter feeding of elk should be discouraged, and no additional public or private feedgrounds be established in the Greater Yellowstone Area. Establishment of emergency or permanent feedgrounds for other wild ungulates, which may act as an attractive nuisance and concentrate elk or bison, is likewise discouraged (GYIBC website)."

Keiter and Froelicher (1993) further stated that, "in Wyoming at least, any effective response to wildlife brucellosis will almost certainly require reduction – if not elimination – of the elk feedgrounds, which will undoubtedly impact elk population numbers and hunting opportunities." These endorsements and the accumulated serologic data tell us that elk do not maintain brucellosis in the absence of feedgrounds (excepting where they commingle with chronically infected bison). Thus, elk management reliant on winter feeding to maintain excessively large populations of elk clearly perpetuates chronically infected elk herds.

Given the polarization and politicization of the brucellosis issue, Keiter and Froelicher (1993) advocated a regional brucellosis control policy based on the principle of risk reduction, not disease eradication. This is to say that brucellosis in elk and bison should be "managed" in a way that minimizes the risk of transmission of disease from wildlife to livestock. Cheville et al. (1993), Thorne and Kreeger (2002), Peterson (2003), and others have provided useful lists of management actions that may reduce prevalence of brucellosis in wildlife and the risk of transmission to livestock.

Removal of livestock from the GYA or grazing only by neutered yearlings would remove the risk of brucellosis infections of concern to federal and state agricultural interests. Both seem unlikely. If we assume no changes in livestock grazing patterns, disease risk management would include 2 components. 1) Limiting or preventing contact of free-ranging elk and bison with cattle during periods when brucellosis abortions (primarily) and parturition (secondarily) occur in elk and bison – a period covering February through mid-July each year [although Jackson bison (Berger and Cain 1999) and elk (Smith 1994a) occasionally give birth and presumably abort outside those months]. 2) Reducing the prevalence of brucellosis in elk and bison herds. Remedies to the chronic infection rates of southern GYA elk either lie in elimination of elk feedgrounds, or reducing prevalence of the disease in elk that use the feedgrounds and are associated seasonally with those animals. Because long-range movements of elk into and out of the western Wyoming and eastern Idaho feedground complex occur, a sizeable area must be considered – one far beyond the scope of the BEMP.

A major hurdle to reducing infection rates of chronically infected elk and bison herds is that no reliable data exist regarding how the probability of intra- or interspecific brucellosis transmission decreases as a function of decreasing *B. abortus* prevalence in bison or elk herds. Peterson (2003) advises that the risk may not decline linearly, or at all, where elk remain concentrated on feedgrounds. A single aborted fetus hosts large numbers of infectious doses of *B. abortus* that could infect large numbers of elk at a feedground (Thorne 2001). The same is true in free-ranging bison because of their gregarious nature (Davis et al. 1995).

Setting this unresolved and problematic issue aside, two *Brucella* vaccines, developed for vaccination of cattle against brucellosis, have been evaluated in elk. Clinical trials using RB51 *Brucella* vaccine in elk were unsuccessful in conferring protection against abortion (Kreeger et al. 2002). After several years of clinical trials of Strain 19 *Brucella* vaccine in elk, the Wyoming Game and Fish Department developed an integrated program in the late 1980s to eliminate brucellosis in feedground elk and to keep elk and cattle separated to prevent *B. abortus* transmission. The Brucellosis-Feedground-Habitat (BFH) Program used ballistic vaccination of feedground elk with Strain 19 vaccine to reduce brucellosis infection rate. Habitat enhancements on winter ranges adjacent to feedgrounds, designed to limit the duration of feeding and elk concentrations, complemented the vaccination program. Vaccinations began in 1985 at the Alpine feedground, and nearly all calf elk at 21 of 22 feedgrounds have been vaccinated on an annual basis beginning in the mid-1980s to mid-1990s. The 22nd feedground, Dell Creek, serves as a control where elk are not vaccinated. Although some initial declines in seroprevalence occurred, the 23.6% collective seroprevalence among vaccinated elk reported during 1993-2004 did not statistically differ from the 32.4% seroprevalence of unvaccinated elk at Dell Creek (Dean et al. 2004). More troubling is that earlier declines in seroprevalence of the Alpine feedground elk reversed in 2000 with seroprevalence ranging from 50-59% during 2001-2004 (Dean et al. 2004). The reason for the return to prevaccination seroprevalence rates are uncertain (Peterson 2003).

Research on efficacy of Strain 19 indicates it confers 25% protection against abortion and no protection against infection under controlled conditions (Roffe et al. 2004). The level of protection that Strain 19 confers to free-ranging elk, given all other factors that can affect disease transmission, variability of exposure dosages, individual susceptibility to disease, and herd immunity, remains uncertain (Gross et al. 1998, Roffe et al. 2004). Gross et al. (1998) modeled brucellosis seroprevalence over a 100-year time-frame under a variety of conditions. Intensive vaccination with a vaccine having a 25% efficacy did not eliminate brucellosis. That modeling predicted a reduction in brucellosis seroprevalence by 40-50% with supplemental actions, such as test and slaughter of seropositive animals, required to eradicate the disease. Roffe et al. (2004) did not recommend the use of Strain 19 for wildlife management because it is "...highly unlikely to lead to significant reduction or eradication of brucellosis in feedground elk". Furthermore, ballistic vaccination of large numbers of elk requires access to the animals that only confinement or feeding permits. This crowding of animals is at the heart of the transmission-infection-disease perpetuation cycle that has persisted in feedground elk.

Boyce (1989) reported brucellosis seroprevalence rates among adult female elk on the NER during 1970-1985 averaged 39%. In the absence of all but a small scale experimental vaccination program by the State of Wyoming during 1989-1991,

brucellosis seroprevalence at the NER averaged 14.7% in recent years (Dean et al. 2004). These data suggest that the much larger numbers of elk fed on the NER, an order of magnitude greater than on state feedgrounds, did not increase seroprevalence. Furthermore, seroprevalence rates were unrelated to elk numbers on the NER since recurrent testing for brucellosis began in 1970 (Smith and Roffe 1997). Smith and Roffe (1997) suggested that the type of feed (or method of feeding) provided elk may have affected seroprevalence of brucellosis, which was lower consequent to the NER's conversion from feeding baled hay to mechanized feeding of pelleted hay. This may occur because feeding with mechanized equipment facilitates spreading elk out across feedgrounds and moving elk to clean areas for feeding. Moreover, pelleted alfalfa is more rapidly ingested and more rapidly digested than long hay. Elk spend less time concentrated on feedlines, and more time distributed across the refuge foraging on standing grasses. In addition, elk are fed fewer days each winter at the NER than at state of Wyoming feedgrounds (Smith 2001, Western EcoSystems Technology 2004). Thus, feed type, feeding method, and the duration of feeding reduce concentrations of animals on feedgrounds where brucellosis is most likely transmitted among elk.

The above discussions suggest three things: 1) crowding of elk on feedgrounds maintains brucellosis in southern GYA elk, 2) time spent on feedgrounds and feedlines increases exposure to and transmission of *B. abortus*, 3) vaccination of elk with Strain 19 is unlikely to reduce seroprevalence rates of feedground elk sufficiently to satisfy the concerns of government administrators and the livestock industry about the risk of brucellosis transmission from elk to livestock.

Although several invasive population management techniques, listed by Thorne and Kreeger (2002), are potentially useful for reducing brucellosis prevalence in southern GYA elk herds, they are unlikely to be accepted by the general public for a variety of reasons, both ethical and practical in nature. Thus, we are left with elimination of elk feedgrounds as the most practical means of greatly reducing brucellosis. Although there is no assurance that this approach will eradicate brucellosis from the southern GYA elk herds, it is certain to be more successful in advancing that goal than the management practices used over the past several decades. Its success is based upon keeping the size of elk herds within carrying capacity of available winter range, which reduces animal to animal contact and exposure to pathogens. As opportunities for disease transmission decline and attrition removes brucellosis infected elk from herds, an increasingly diminished infection rate emerges.

Regarding the Jackson elk herd, winter mortality would increase in the absence of winter feeding (Hobbs et al. 2003). Also, a smaller number of elk wintering on the NER would produce a smaller annual surplus to be removed, alleviating the necessity of long hunting seasons on the NER and southern GTNP. Consequently, elk may not vacate transitional and early-season winter ranges in GTNP and the northern NER to congregate on the southern half of the NER (which is closed to hunting) beginning in October. Furthermore, elk would graze in smaller group sizes across the NER, southern GTNP, and adjacent National Forest lands during winter, when they are no longer reinforced by the prospect of being fed to congregate in large numbers on the south half of the NER 2-3 months before winter feeding commences. Termination of feeding would yield important benefits for refuge habitat conditions and other game and nongame wildlife species (Smith et al. 2004).

Carrying capacity of winter ranges in Jackson Hole varies markedly with environmental conditions, particularly growing season weather conditions and winter severity (Hobbs et al. 2003). Natural and human-caused changes to habitats modify habitat effectiveness (Toweill and Thomas 2002). If feeding were phased out at the NER, elk that winter on the NER would number fewer than in recent years, and numbers throughout the Jackson herd unit may fall below the 11,029 objective number currently authorized by Wyoming Game and Fish Department (USDI 2005a:79, 253).

To increase the chances of successfully weaning elk that migrate to the NER from winter feeding, managers must be willing to accept a winter herd size nearer the number the refuge can sustain in a severe winter than the number sustainable in an average or mild winter. Through adaptive management, if the modeled population sizes for various winters (Hobbs et al. 2003) prove too low, population sizes can be revised upward. The pace at which feeding of elk on the NER could be phased out is dependent on the pace at which herd reductions to habitat carrying capacity can be effected. Secondly, should a decision to phase out feeding ensue from the BEMP-EIS process, feeding should not be terminated until the elk herd size is reduced to winter range carrying capacity. Premature termination of feeding, while elk and bison numbers exceed winter habitat capacity, could result in unacceptable winter losses and many animals moving onto private lands in search of forage.

Finally, the Wyoming Game and Fish Department (Clause et al. 2002) and a private consultant (Western EcoSystems Technology 2004) identified the 3 feedgrounds in the Gros Ventre drainage among those with the highest potential for moving elk off feedgrounds and onto native winter ranges. Wildlife telemetry studies (Smith and Robbins 1994, Smith 1994b) and observations of neckbanded elk demonstrate some interchange of elk between the NER and Gros Ventre feedgrounds. Thus, if an effort to phase out feeding on the NER were coordinated with a like action in the Gros Ventre, the potential for elk that have been fed on the NER to find and habituate to the Gros Ventre feedgrounds could be avoided. A coordinated State-federal effort would serve to wean the Jackson elk herd from winter feeding.

Bison

As previously noted, bison herds can maintain chronic infections of brucellosis in the absence of winter feeding or other forms of artificial crowding, suggesting that like cattle, bison are a more natural host for *B. abortus*. Their gregariousness, including during parturition, provides ample opportunity for exposure to infected fetuses and live births (Cheville et al. 1993). Thus, the elimination of feeding on the NER is highly unlikely to eliminate brucellosis in bison, although there may be some decline in the level of infection (URS 2003). The likelihood of Jackson bison to remain chronically infected presents two problems: 1) bison would be a continuing source for transmission of *B. abortus* to cattle, and 2) bison would be a continuing source for reinfection of elk. Thus, unlike elk from which brucellosis could be expected to decline to very low prevalence over time once weaned from feedgrounds, bison would require more rigorous management.

The Jackson herd remains sufficiently small and confined in distribution that an effective vaccination program could be undertaken. Both Strain 19 and RB51 *Brucella*

vaccines have been administered to bison in clinical trials to protect bison against *B. abortus* induced abortions and to protect against infections. Strain 19 vaccinations of both pregnant adult bison and bison calves induced abortions in 57% of adult vaccinated bison and 73-83% of calfhood vaccinated bison. Strain 19 conferred no protection against abortion or infection (Davis et al. 1991, 1993). Peterson's (2003) review of the RB51 vaccine trials concludes that it offers little protection against abortion or infection in bison.

Strain 19 and RB51 vaccines were developed in the mid-20th Century to protect cattle against brucellosis. The clinical trial results referenced in this report show that elk and bison immunologically do not respond to these vaccines as cattle do. The chances are not good that a new vaccine for bison or elk will be developed in the near term. Without sufficient profit motive, American drug manufacturers are reluctant to conduct the research, development, testing, and marketing of vaccines against virulent human pathogens (e.g. Asian bird flu), which have far larger sales markets. Unless a persistently low prevalence of brucellosis in elk is tolerable, bison will require additional management applications, beyond termination of winter feeding. Otherwise, elk may become reinfected by contact with bison -- one explanation for the chronic brucellosis infection of elk (albeit at low prevalence) on the Northern Range of Yellowstone National Park (Cheville et al. 1997). In short, an integrated approach to brucellosis reduction/elimination on the NER and GTNP will require effectively addressing brucellosis in both elk and bison to be successful.

CHRONIC WASTING DISEASE

The Disease

Chronic Wasting Disease (CWD) is one of a group of fatal, transmissible spongiform encephalopathies (TSEs) that affect the central nervous system of a range of mammals, including humans. Scrapie, which affects domestic sheep, bovine spongiform encephalopathy (BSE or mad cow disease) which affects cattle, and Creutzfeld-Jacob disease (CJD and vCJD), which affects humans, were probably among the best known of the TSEs, until CWD became widespread in North American farmed and free-ranging cervids over the past decade. The best evidence suggests that all TSEs are caused by prions, non-DNA containing proteins. A remarkable characteristic of these non-living proteins is that they are highly resistant to environmental degradation, and can be indirectly transmitted to other animals through excreta, contaminated soil, and decomposing carcasses, as well as by direct animal to animal contact (Williams et al. 2002, Miller et al. 2004). CWD is apparently not a food-borne disease as was the case with BSE (Williams et al. 2001). There is no known immune response to the CWD agent and no immunization or cure for this fatal disease. Williams et al. (2001) and Williams et al. (2002) provide reviews of CWD and related TSEs.

Distribution

CWD was first recognized in captive mule deer and elk in research facilities in Colorado and Wyoming (Williams and Young 1980). The disease was subsequently

found in free-ranging mule deer, elk, and white-tailed deer in northcentral Colorado and southeastern Wyoming during 1985-1990. The origin of the CWD causing prion and whether the disease originally arose in cervids in research facilities or in the wild is unknown. The rapid spread of CWD among cervid game farms was likely facilitated by transport of diseased deer and elk among game farms. CWD infected elk have since been found in game farms in Colorado, Kansas, Minnesota, Montana, Nebraska, Oklahoma, South Dakota, Wisconsin, Alberta, Saskatchewan, and South Korea. Infected captive deer and elk herds are depopulated or quarantined once identified (Williams et al. 2002). Carcasses are generally disposed of by incineration or burial.

Since 2000, CWD has spread from infected elk farms to wild cervids in Saskatchewan, Nebraska, and South Dakota. The origins of CWD in free-roaming elk in northwestern Colorado, white-tailed deer in Wisconsin and Illinois, and mule deer in New Mexico and Utah remain unclear. Most recently, CWD was discovered in free-ranging mule deer in Alberta, white-tailed deer in New York and West Virginia, and a moose in Colorado –the first wild moose and the fourth cervid species diagnosed with CWD. CWD has not been demonstrated in bison or other bovids.

Closer to Jackson Hole, CWD has spread to wild cervids across the northern third of Colorado and as far south as Colorado Springs. In 2002, CWD infected mule deer were diagnosed near Vernal, Utah, some 200 miles south of the NER. CWD has progressed northward and westward in Wyoming from the endemic area of southeastern Wyoming, and new areas of infection annually have been detected by surveillance (Wyoming Game and Fish Department website). Mule deer infected with chronic wasting disease were found near Baggs, Wyoming, just west of the Continental Divide, and in fall 2003 near Worland, Wyoming, just 90 miles east of the Jackson elk herd unit. Then in October 2005, two harvested mule deer tested positive for CWD immediately northwest of Thermopolis, Wyoming (Wyoming Game and Fish Department website). CWD has not been identified in deer or elk in Idaho.

Surveillance and Prevalence

CWD is transmissible among mule deer, white-tailed deer, and elk, and free-ranging populations of these species potentially link the most recent cases of CWD infection in Wyoming, or others that have remained undetected, with deer and elk west of the Continental Divide. The state of Wyoming initiated surveillance for CWD in 1997 by sampling and examining tissues from hunter killed deer and elk. In the endemic area of southeastern Wyoming, the overall prevalence found in harvested deer and elk have averaged 7.7% and 3.4%, respectively (Dean et al. 2004). Prevalence appears to run higher in white-tailed deer than mule deer, and latency of the disease appears to be shorter in white-tailed deer, as 5-6 month-old clinically ill fawns have been reported in Wisconsin and Nebraska. The youngest free-ranging mule deer and elk detected with CWD were ≥ 17 months of age (Samuel et al. 2003).

The Wyoming Game and Fish Department initiated sampling of elk from the Jackson elk herd for bovine tuberculosis (TB) in 1992. Again, samples were collected from harvested elk, primarily from GTNP and the NER. Beginning in 1996, tissues were also collected from most of these harvested animals to conduct surveillance for CWD. Through 2002, 2,532 elk were tested for TB and 1,095 for CWD (Dean et al. 2004). No

positive test results have been obtained for either disease to date (Wyoming Game and Fish Department web site). In addition, NER staff has recorded all elk mortalities that occur on the refuge for many years. Information about animal health and factors that contribute to mortality are recorded and necropsies performed on suspect mortalities that may harbor non-endemic diseases (Smith and Roffe 1994). No CWD mortalities have been documented.

Potential Threat to Jackson Elk

Potential sources of infection of Jackson elk include game farmed cervids or free-ranging cervids infected with CWD, or infectious materials from dead animals transported to Jackson Hole that are discarded on the landscape. Disease has not been reported in the nearest game farms in eastern Idaho. With stricter regulation of interstate transport of cervids from and to game farms, the opportunity for clinically or subclinically infected animals to enter the three elk farms in eastern Idaho, or other game farms near the GYA, is much reduced from just a few years ago. A growing number of states have adopted regulations prohibiting the transport of carcasses, or portions thereof most likely to harbor disease causing prions, out of known CWD infected areas. The movement of live animals is also strictly regulated by most states to avoid the import or export of CWD infected animals.

If we consider possible sources and routes by which Jackson elk may become naturally infected with CWD, we are dependent on understanding the true regional distribution of CWD among cervids, movement and seasonal distribution patterns of individual populations, and potential for transmission of CWD among herds based upon duration and seasonality of shared ranges.

Detection of CWD Distribution

It is highly unlikely that the total distribution of CWD in wild cervid populations is known. First, there is a lengthy incubation period in mule deer and elk (Williams et al. 2002), and secondly practical limits to the intensity and extent of sampling of CWD in Wyoming and elsewhere results in a probability of detection of CWD infected animals that is low where population sizes are high, sampling intensities (harvest rates) are low, or disease prevalence is very low. For example, Wyoming collected tissues from 3,958 deer and elk harvested statewide in 2004 for diagnostic testing. The goal was to provide a 99% probability of detecting CWD if it existed at a $\geq 1\%$ prevalence in each of the state's seven administrative regions. Statewide population estimates were >92,000 elk, >500,000 mule deer, and an undetermined number of white-tailed deer in Wyoming in 2003. Some 35,000 of the state's mule deer, or about 7%, were harvested in 2003 (Wyoming Game and Fish Department 2004). I crudely estimated 147,000 white-tailed deer throughout Wyoming by applying this same 7% harvest rate to the 10,328 white-tails harvested in 2003. Thus, the estimated statewide population of deer and elk totals some 740,000 animals, necessitating a massive sample collection effort to meet the above standards (99% probability of detecting CWD if it existed at a $\geq 1\%$ prevalence) and requirements of random sampling established for surveillance of CWD in free-ranging cervids (Samuel et al. 2003).

Prevalence was generally $\geq 1\%$ of elk and deer herds in Colorado and Wyoming before sampling of harvested animals detected newly infected areas or populations (Miller et al. 2000). A 99% probability of detection of at least one infected animal in a herd of 10,000 deer or elk having a $\geq 1\%$ infection rate (≥ 100 infected animals) would require collecting tissues from 449 animals, a number greater than was achieved (104-317 sampled annually) during each year of the 5 years of CWD sampling during 1996-2002 of the Jackson elk herd. The level of sample collection in one of those 5 years (317 in 1998) would have provided a 99% probability of detecting a 2% or greater infection rate. However, 2% of a population of 10,000 represents ≥ 200 infected animals – a level of infection that likely could sustain itself where animals are seasonally highly concentrated. Thus, when the first handful of CWD infections occurs in a large deer or elk herd, the probability of detection through random hunter field checks or check station collections is unfortunately quite low.

Without complete depopulation and testing, failure to detect CWD does not translate into absence of the disease (Samuel et al. 2003). Areas with CWD positive deer and/or elk have likely gone undetected in Wyoming and elsewhere. This is one reason that Wyoming, Colorado, Wisconsin, and other jurisdictions continue to detect newly infected herds/locations each year. The other reason for geographical expansion of CWD is that deer and elk are highly mobile and dispersing individuals, that may infect animals in adjacent herds, are periodically sampled.

Certainly, Wyoming and other states have finite resources that they can expend on CWD surveillance, and Wyoming did not conduct statewide surveillance for CWD until fall 2003. But this means that CWD infected cervids in areas such as the Gros Ventre drainage or areas in and adjacent to the Green River basin's feedground complex would presumably only have been detected prior to 2003 if tissues from a clinically ill animal were collected. Such focused surveillance of sick animals by trained agency personnel, often alerted by contacts from the public, is termed targeted surveillance. Targeted surveillance increases the probability of detection of diseased animals, but this is not a systematically conducted procedure in most states (Samuel et al. 2003). Rather it is an opportunistic identification of animals presenting the clinical signs of CWD – which in and of themselves are not diagnostic of CWD. Laboratory testing by established procedures is still required to diagnose disease. More importantly, Miller et al. (2000) noted that targeted surveillance was a poor predictor of CWD prevalence in an area or population, only a means of detection. There are no antemortem tests for CWD other than tonsillar biopsy, which is only useful in deer and is impractical and expensive in a wildland setting (Wolfe et al. 2002).

Movements of Cervid Populations

Western Wyoming is home to the largest and most diverse ungulate populations in the Rocky Mountain region ((Sawyer and Lindzey 2001), including tens of thousands of elk, mule deer, pronghorn antelope, and thousands of white-tailed deer, moose, and bighorn sheep, as well as mountain goats. As described in the overview, the Jackson elk and bison herds interchange or seasonally share ranges with elk herds to the north, south, east and west. Moreover, mule deer, white-tailed deer, and moose herds, some of which are highly migratory, occur within and adjacent to the Jackson elk herd unit.

Recent studies of the 30,000 – 40,000 strong Sublette deer herd, which winters in the Green River basin near Pinedale, demonstrate that these deer migrate 60-100 miles to the east, west and north and summer in five mountain ranges (Sawyer and Lindzey 2001). This includes the Gros Ventre Range, which also provides summer range to elk that winter on the NER, 3 Gros Ventre feedgrounds, and adjacent winter ranges on national forest lands. In addition, some mule deer and white-tailed deer from the Dubois Herd Unit, east of the Continental Divide, and Jackson cervids overlap/interchange in the upper Gros Ventre drainage. Some 60% of radiocollared adult female elk leave the Wiggins Fork Herd Unit, near Dubois, during summer and fall (Queen and Ryder 1996). Some of those also spend summer-fall along the Continental Divide of the Gros Ventre Range and the Teton Wilderness.

Although the Jackson elk herd may be the most studied elk herd in North America (Boyce 1989), only recent investigations (2002-2003) using radio-telemetry revealed that 32% of female elk wintering in the Gros Ventre drainage may spend all or part of the summer-fall seasons in the Green River drainage or east of the Continental Divide in the Wind River drainage – watersheds occupied by the Green River and Wiggins Fork elk herds, and the Sublette and Dubois deer herds (Figure 1). CWD is not known to occur in Idaho. Therefore natural expansion of the disease is likely to occur from Wyoming source cervid herds. The most likely routes by which CWD may enter the Jackson elk herd are from the east from the Dubois area, or from the Green River basin to the south. Diseased animals would likely first appear in the Gros Ventre drainage, the eastern and southern hydrographic divides of which serve as migratory pathways. The most recently detected expansion to Worland and then Thermopolis, Wyoming, puts CWD at the foot of the Owl Creek and Absaroka Mountains, ranges that rise to the west toward the Continental Divide and support large populations of deer and elk contiguous with the Jackson elk.

Infected deer or elk moving into the Jackson elk herd, or alternatively, elk and/or deer from Jackson commingling with infected deer or elk to the east or south, then returning to Jackson Hole, could initially bring the disease to Jackson Hole. This of course would require some unknown level of prevalence to occur in the Dubois or Green River areas, likely as a result of natural expansion of known disease foci near Worland/Thermopolis and southcentral Wyoming, or as yet undetected locations farther west. If and when this will occur cannot be predicted. However, prevalence of CWD has been stable or increased and geographic distribution continued to expand among free-ranging deer and elk populations (Miller et al. 2000, Miller and Conner 2005, Wisconsin Department of Natural Resources website). Mountainous topography, rivers, and other geographic barriers have not prevented the spread of CWD in Colorado, where the disease has been most intensively studied in free-ranging deer and elk. Well-documented movements of deer and elk into and out of Jackson Hole and the sharing of portions of seasonal ranges with adjacent herds provide little comfort that the Jackson cervid populations are somehow isolated from the outside world.

Why Concern Over CWD in Jackson Hole

Concerns over the introduction of CWD into the NER and western Wyoming's feedground elk in general are many-fold.

1. The conditions of animal crowding, shared feedsites, bedsites, water sources, and accumulated excreta on feedgrounds promote relatively unsanitary conditions that benefit many pathogens and promote transmission of diseases which have a density dependent component. This explains why brucellosis is maintained in feedground elk, but not those unassociated with feedgrounds or other infected species. Prevalence of CWD in research facilities has exceeded 90% (Williams and Young 1980). Peters et al. (2000) reported prevalence of CWD in game farmed elk of 59%, although few prevalence rates among game farmed elk have been reported. Furthermore, infected captive herds are generally destroyed soon after discovery of CWD so that prevalences of protracted epidemics are not achieved.
2. CWD will amplify on feedgrounds because of increased animal-animal contact.
3. Environmental loading of feedsites with infectious amounts of the CWD agent will occur.
4. Animals return to feedgrounds year after year (Smith and Robbins 1994, Smith 1994b) with recurrent opportunities for direct or indirect infection.
5. Probability of infection increases with age, at least in deer (Miller and Conner 2005), and feedground elk are long-lived (Boyce 1989, Smith and Anderson 1998, Clause et al. 2002).
6. Modeling 1.2-1.3 infectious contacts per infectious individual produced the most plausible disease and population dynamics in mule deer. When horizontal transmission rates exceeded 1.3, rates of transmission rapidly exceeded those observed to date from field data (Miller et al 2000). Much higher transmission rates must occur in captive cervids to explain observed epidemics of confined populations (Miller et al 2000).
7. Models forecast that when CWD epidemics reach 5% prevalence, steep declines in populations could occur over 30-50 year time scales without management intervention (Gross and Miller 2001).

Miller and Conner's (2005) recently published paper on CWD in mule deer of Colorado makes the following additional points that should be of interest to natural resource managers of western Wyoming.

1. Based on documented prevalence, spatial distribution, and computer modeling, their findings reinforced previous work (Miller et al. 2000) that CWD among cervids in the endemic areas of northeastern Colorado and southeastern Wyoming represents an epizootic with a protracted time scale. That is, the disease outbreak is still in the early stages and will continue for some unknown time.
2. Disease prevalence increased in both sexes over a 7 year time span. Prevalence of CWD was higher on winter ranges within a game management unit than throughout the game management unit. They state the "data suggest that

areas where deer congregate seasonally may be particularly important in sustaining CWD epidemics in free-ranging populations.”

This latter point is both good news and bad news for those who may advocate proactively addressing CWD before it affects the Jackson elk herd and the Wyoming feedground complex in general. The bad news is that even in free-ranging populations of mule deer in Colorado, where CWD is established, CWD epidemics may be sustained by seasonal concentration of animals. The good news is that if concentrations of animals can be reduced by population reductions and by enhancement of habitats and animal distributions, managers may be able to limit disease transmission and prevalence.

Williams et al. (2001) noted that control of CWD is problematic in captive cervids and more so in free-ranging cervids. The potential number of susceptible hosts, their wide distributions and movements, the ecological and economic values of these resources, and legal and ethical constraints on treating public resources the same as captive private herds are among the difficulties of testing and controlling the disease in wildlife. Feedgrounds probably represent conditions intermediate between free-ranging populations and herds that are confined yearlong. Elk remain on the NER about 6 months/year (Smith and Robbins 1994, Smith et al. 2004). Animals are fed an average 2 months/year and longer on state feedgrounds (Smith 2001, Western Ecosystems Technology 2004), at which time functional densities may exceed those observed in game farms (Peterson 2003, USDI 2005). Feces accumulate to depths on feedgrounds that require harrowing each spring to break up this mat of organic material that suppresses growth of new grasses. *B. abortus* and other pathogens may remain viable for 100 days in such pastures (Thorne and Kreeger 2002). Some CWD epidemics have been attributed to contaminated pastures, suggesting prions persist in contaminated environments for >2 years (Williams et al. 2002, Miller et al. 2004).

Without better information about CWD transmission mechanisms, seasonality of transmission, and the relative risk of indirect transmission (via environmental contamination) versus direct transmission, one cannot quantitatively predict CWD prevalence that feedground elk may achieve compared to game farmed elk. However, if prevalence is even half that of game farmed elk (Peters et al. 2000), an escalating rate of CWD-induced mortality will drive populations toward extinction, based upon modeling of the disease in mule deer (Miller et al. 2000, Gross and Miller 2001). As Smith (2001) noted, should a disease as virulent and transmissible as CWD or bovine tuberculosis become established within the GYA, the number of infected herds of susceptible species could rapidly expand. Twenty-five elk herds alone, totaling 120,000 elk, winter in the GYA (Toman et al. 1997). Because distributions of adjacent herds overlap, disease could ultimately spread across the 18 million acre area. In Jackson Hole or the GYA in general, public alarm and outrage over the expected ecological and socioeconomic impacts will compel elected officials, resource managers, and disease regulators to take drastic actions long before elk populations are in jeopardy. However, once well-established, our current understanding and available tools are insufficient to eliminate CWD, short of depopulation.

In general, it is much easier to prevent the introduction of a disease into a given area than to control or eradicate a disease that has become established (Wobeser 1994). Surveillance is important for detecting new foci of CWD, assessing spatial distribution

and prevalence, and monitoring changes over time (Samuel et al. 2003). However, surveillance must be complemented with actions that reduce the risk of CWD spreading to new locations, and limit amplification of CWD in newly infected herds to effect management programs that protect populations. Amplification addresses the potential for increased transmission rates, prevalence, mortality, and resultant ecological and socioeconomic impacts consequent to a previously disease-free population or area experiencing its first case(s) of a new disease. Risk factors associated with both exposure of wildlife to CWD and amplification of CWD following exposure appear in Table 1.

Table 1. Known and suspected CWD risk factors (from Samuel et al. 2003).

Exposure Risk Factors	1) Areas adjacent to CWD-positive wildlife 2) Areas adjacent to land on which TSE-positive animals, farmed or wild, have lived 3) Areas with CWD-positive farmed or captive herds 4) Areas with concentrations of farmed or captive elk or deer 5) Areas that have received translocated deer or elk from CWD-infected regions 6) Areas permitting transport of hunter-killed elk or deer carcasses from CWD infected areas
Amplification Risk Factors	1) Areas with high elk or deer population density 2) Areas with a history of CWD animals or CWD contaminated environments 3) Areas with low abundance of large predators 4) Areas where free-ranging elk or deer are artificially concentrated by baiting, feeding, water development, and other human related habitat modifications

The exposure risks of NER elk, or more generally cervids of western Wyoming, contracting CWD include risk item 6. The lack of game farms in Wyoming is a positive. The nearest known locations of CWD infected wild animals are 90 miles west of the Jackson elk herd unit. Wyoming adopted a regulation in 2005 prohibiting the transport and disposal of carcasses, or parts thereof, from the CWD endemic area to other areas within and outside Wyoming (see Wyoming Game and Fish Department website).

Amplification risk factors for NER elk, and western Wyoming elk in general, include 1 and 4. These are significant risk factors as the scientific literature is replete with information that high cervid densities and artificial concentrations encourage transmission and amplification of disease, including CWD (see Dunkley and Cattet 2003). Large predators remove sick and otherwise compromised individuals from prey populations, limiting exposure of conspecifics and other susceptible species to disease.

The Wyoming Game and Fish Department has primary responsibility for management and health of the state's resident wildlife. Although Wyoming's 2005 draft Chronic Wasting Disease Plan discusses elk feedgrounds, the plan does not prescribe proactive management that will limit amplification of CWD in western Wyoming.

Section 11, Disease Management, of the draft plan acknowledges that CWD may reach northwestern Wyoming, and that much higher prevalence rates and mortality may occur in feedgrounds than in non-fed elk, based upon research from captive elk. Yet the plan prescribes no actions to reduce elk populations and the feeding practices that crowd animals onto feedgrounds. This is particularly troubling when the same section acknowledges that “CWD may reach NW Wyoming” and “the spread of CWD, at best, can be slowed, but not prevented.” A number of states have implemented programs to reduce densities and ban private feeding of cervids. These include Colorado, Montana, Nebraska, and Wisconsin which do not have state sponsored cervid feeding programs. In 2003, Teton County and the Town of Jackson, Wyoming adopted citizen-sponsored bans on private feeding of wild ungulates.

The only actions the draft plan prescribes for reducing feedground elk densities or feeding practices are retroactive – after CWD is discovered on feedgrounds. Furthermore, those 3 prescriptions have been in existence since Wyoming’s BFH program was adopted in the late 1980s, and have failed to reduce elk numbers on feedgrounds or the duration of feeding (Western EcoSystems Technology 2004).

As the BEMP points out (USDI 2005:136), “the US Fish and Wildlife Service and National Park Service can do little to prevent Jackson Hole mule deer and elk from contracting chronic wasting disease from other ungulates outside the Jackson elk herd unit and transporting it into Jackson Hole.” However, Williams et al.(2002) conclude, “CWD could have a dramatic influence on management of free-ranging cervid herds where it is present. Responsible wildlife management and animal health agencies must act to limit distribution and occurrence of CWD in free-ranging and farmed cervids ...”

In anticipation of CWD arriving at some point in the future, and with no assurance of better technological means of eliminating CWD than we have for brucellosis or a variety of other cervid diseases (Peterson 2003), the NER and GTNP can proactively adopt prudent measures that will limit the amplification of CWD. 1) Reducing population density is a recognized method for disease control and is based on the idea that infectious disease is density dependent (Wobeser 1994). Population reduction may be used in areas in which CWD is not yet present, with the aim of reducing the potential for the disease to become established or to amplify (Chronic Wasting Disease Taskforce 2002). 2) Phasing out the winter feeding program will limit disease transmission and prevalence in the NER and GTNP (see Van Deelen 2003, Peterson et al. 2002, Dunkley and Cattet 2003). This second measure is dependent upon the first because elk are fed at the NER due to insufficient winter habitat and forage to support current numbers (core problem identified in USDI 2005).

Although CWD is not a disease of bison, it is unreasonable to discontinue winter feeding of elk but not bison. Just as bison discovered the NER elk feedgrounds (Peterson 1991), elk will habituate to areas where feeding is intended only for bison. Thus, the Jackson bison herd will also need to be maintained within winter range carrying capacity. Both actions, population size adjustments and phasing out of winter feeding, should be accompanied by multi-agency programs carried out to enhance the carrying capacity of winter and transitional ranges, as has been laid out in the Jackson Interagency Habitat Initiative. Aune et al. (2002) provided examples of habitat-based programs that conserve elk without winter feeding. A multi-jurisdictional, multi-species approach would best accomplish disease management goals (Chronic Wasting Disease Taskforce 2002,

Peterson et al. 2002). In concert, the above measures will limit direct and indirect routes of transmission, should CWD arrive in Jackson Hole.

Environmental contamination with the infectious agent is a particularly insidious characteristic of CWD where cervids are crowded. It would serve as a perennial source of CWD exposure as elk return to feedgrounds each winter. Infectious agent accumulation would likely vary on a gradient from highest on feedgrounds to lowest on high elevation summer ranges. How much contamination is required on native ranges to provide infectious doses is unknown, but is likely a function of animal density. Without feeding, environmental contamination sufficient to cause disease may still occur in some areas (Miller and Conner 2005), but likely be far less than on feedgrounds where functional densities are far greater. The agent is extremely resistant to chemical disinfectants as well as to physical methods of inactivation. It is still not known whether environments contaminated with TSE agents can ever be completely disinfected (Williams et al 2002).

OTHER DISEASES

Peterson (2003), URS (2003), and the 2002 panel of disease experts identified a number of macroparasitic and microparasitic diseases of concern that currently do or potentially could infect the Jackson herds. URS (2003), based upon opinions of the 2002 panel, evaluated and ranked the relative impact and environmental consequences of these diseases becoming established in the elk and bison herds. Rather than reiterate, I refer the reader to those sources for thorough analyses of diseases. Instead, I will highlight information on three of the endemic diseases among refuge elk and briefly summarize discussions about two virulent, undocumented diseases.

Endemic Diseases

The same crowded conditions responsible for high prevalences of brucellosis in elk and bison on feedgrounds can also foster the transmission and maintenance of other density-dependent diseases. Murie (1951) suggested a link between the prevalence of both scabies and necrotic stomatitis and the overstocked winter ranges in Jackson Hole. Before 1950, Murie (1951) considered necrotic stomatitis to be the most significant cause of winter mortality of Jackson elk. Provisioning of higher quality hay alleviated much of the problem which is caused by entry of the ubiquitous microparasite *Fusobacterium necrophorum* into lesions of the soft tissues of the mouth. Stiff-awned hay and coarse browse associated with overstocked and heavily browsed ranges are primary causes of the lesions. Although cases of necrotic stomatitis still occur on and adjacent to the NER and state feedgrounds, in recent years mortality from this disease has been limited to very few animals. Necrotic stomatitis has not been observed in Jackson bison. Peterson (2003) notes that if winter feeding is curtailed at the NER, managers should maintain elk numbers within winter range carrying capacity to avoid a potential increase in prevalence of necrotic stomatitis.

Psoroptic mange, or scabies, is a condition caused by mites of the genus *Psoroptes*. Severe infestations of these ectoparasites cause a severe exudative dermatitis and alopecia that results in loss of insulation, increased grooming activity, weight loss,

and possibly immune suppression, that predispose severely infested individuals to mortality during periods of severe cold (Smith 1985, Samuel et al. 1991). About 65% (20-30 individuals) of adult male elk that die on the NER annually were afflicted with scabies (Smith 1998). Another 5% of mature bull elk that survive winter show clinical signs of scabies. Scabies reduces survival of trophy size bull elk and the quality of capes of harvested animals. It is unclear whether elk serve as a reservoir for infection of sympatric (geographically overlapping) bighorn sheep (Lange 1982, Peterson 2003). What role feedgrounds may play in transmitting the disease is unknown, although high animal densities would seem to promote transmission. While other factors are probably important in clinical expression, the high density of breeding males in GTNP may play a significant role in the development of scabies (Smith 1985, Smith and Roffe 1994). Reports of psoroptic mange in elk outside the GYA are rare. Psoroptic mange does not occur in bison (Peterson 2003).

Another endemic disease of NER elk, also reported on some Wyoming feedgrounds, is septicemic pasteurellosis. Murie's (1951) single paragraph about this disease (which he referred to as hemorrhagic septicemia) does little more than substantiate that the disease has occurred at the NER for decades. Bison in the GYA have suffered epidemics of this disease, which affects a variety of animals (Miller 2001). Epidemics of pasteurellosis were a significant cause of declining bighorn sheep abundance throughout western North America (Miller 2001). Recently, pasteurellosis was documented during winters 1985-86 and 1986-87 on the NER (Franson and Smith 1988). During the protracted winter of 1992-93, an estimated 160 elk died of septicemic pasteurellosis on the refuge (Wilson et al. 1995). This bacterial (*Pasteurella multocida*) disease is transmitted by direct contact between animals and via aerosols (the exhalants of respiration). It can be particularly virulent among immunologically naïve (not previously exposed to the pathogen) or compromised animals, with rapid progression of clinical signs leading to death (Franson and Smith 1988). It is unknown if the strain of *P. multocida* harbored by Jackson Hole elk threatens sympatric bighorn sheep populations (Peterson 2003). Pasteurellosis in wild ungulates is somewhat dependent on host density (Miller 2001). Peterson (2003) suggests that reducing Jackson elk to winter range carrying capacity and ending winter feeding probably would reduce the incidence of septicemic pasteurellosis in the herd.

Undocumented Diseases

None of the documented diseases of the Jackson elk and bison herds represent the level of population risk that CWD and certain other diseases pose. Undocumented diseases that ranked of high concern among the disease expert panelists, URS (2003), and likewise by Peterson (2003) were bovine tuberculosis (TB) and bovine paratuberculosis (Johne's disease).

Bovine Tuberculosis

Bovine TB is a chronic bacterial disease with a worldwide distribution. Most mammals, including wild and domestic ruminants and humans, are susceptible to the disease (Clifton-Hadley et al. 2001). Bovine TB is caused by the bacterium

Mycobacterium bovis and spreads intraspecifically and interspecifically via aerosols or consumption of contaminated feed. The disease is typically chronic and fatal. Bovine TB has an extended incubation period and the disease may be present in herds long before it is detected as was the case with captive cervid herds in the early 1990s (Clifton-Hadley et al. 2001). Herds of at least 6 species of captive ruminants, including elk and bison, in dozens of game farms across 4 Canadian provinces were diseased and subsequently destroyed. The disease may have been present without detection for 10 years (Roffe and Smith 1992). Elk herds in 8 states (including Montana, Colorado, and the Dakotas) were also part of the infected game farm network. Bovine TB is of major concern to the livestock industry, rebounded in the cattle industry during the game farm epidemics, but the disease is nearly eradicated now from cattle herds (Demarais et al. 2002). Depopulation eliminated bovine TB from captive cervid herds in the U.S. until 2001 when an elk in an eastern Oregon game farm was diagnosed with the disease (Oregon Department of Fish and Game website).

Currently, wild bison in northern Canada and white-tailed deer across much of Michigan's Lower Peninsula are chronically infected with bovine TB. The gregarious nature of both cattle and bison leads to high functional densities of susceptible hosts. White-tailed deer in Michigan have been able to maintain TB due to high densities of deer and the practices of deer baiting during hunting seasons and winter feeding by private citizens (Schmitt et al. 1997). Prevalence among bison tends to be much higher than in deer or elk (URS 2003, Peterson 2003). Surveillance and testing of 2,532 elk in Jackson Hole during 1992-2002 produced no positive cases of TB (Dean et al. 2004).

It was the opinion of the disease expert panel that if Jackson elk and bison herds contract bovine TB in the future, high winter densities compounded by winter feeding would increase transmission and prevalence and TB would be maintained in the herds (URS 2003). Functional densities of elk and bison on the NER far exceed those of deer in Michigan. Should one species contract the disease, it would be spread to the other on the NER feedgrounds (Peterson 2003). As Peterson (2003) noted, "if one desired ideal circumstances for maintaining *M. bovis* in a free-roaming elk population, they would have to go no further than the National Elk Refuge and other GYA feedgrounds." He outlined sweeping measures that would be necessary to eradicate the disease should it become established in Jackson elk and bison, including drastic reductions in elk numbers and test and removal of bison. These measures would be necessary, in combination with elimination of winter feeding, to not only eradicate the disease in Jackson Hole, but to limit its spread elsewhere in the GYA (Peterson 2003).

Bovine Paratuberculosis

Bovine paratuberculosis, or Johne's disease, is caused by *Mycobacterium paratuberculosis* and is a disease of ruminants world wide. *M. paratuberculosis* is related to *M. bovis* and like bovine TB, it produces chronic disease with a long incubation period. The majority of animals in an infected herd may never develop the clinical signs of rough hair coat and gradual loss of body condition, but continue to shed *M. paratuberculosis* in feces (Williams 2001). Susceptible animals ingest the bacteria while feeding or drinking and host density and environmental contamination play a significant role in the shedding-transmission-infection cycle. This is a disease of a significant and

growing concern among livestock interests. Johne's disease can cause mortality in cattle herds ranging from 1-25% (Williams 2001). Population effects, should the disease develop in either Jackson elk or bison, are not certain. Increased density of bison and elk on the NER and winter feeding would enhance disease transmission (Demarais et al. 2002), and therefore prevalence, and mortality (URS 2003).

The disease has been documented in captive and free-ranging elk herds in North America and in ranched bison in the northern GYA, but not in the Yellowstone National Park bison (Peterson 2003). Like bovine TB, the potential sources of contact with Johne's disease for Jackson Hole elk and bison would be infected cattle or farmed cervids or bison. Preventing the introduction of either *M. paratuberculosis* or *M. bovis* through any of these ruminant sources should be a high priority for managers in Jackson Hole (Williams 2001, Peterson 2003).

EFFECT OF ALTERNATIVES ON BRUCELLOSIS AND CWD

The focus of my comments, as requested by the GYC, is to explore and discuss the relationship between disease and feeding of elk and bison. Only two alternatives in the BEMP, Alternative 2 and 6, prescribe a phase out of winter feeding. All others maintain the status quo or some diminished frequency/duration of feeding of elk and bison on the NER with concomitant adjustments in elk and bison population sizes. Alternative 4 is the preferred alternative of the BEMP. Alternative 6 is the alternative endorsed by the GYC.

I was asked to compare how disease impacts would differ between alternatives 4 and 6, the principal elements of which appear in Table 2. Although comparisons of the BEMP's alternatives were previously made by the disease expert panel and by URS (2003), those evaluations are not specifically useful as the BEMP's alternatives were reconfigured subsequent to those efforts. Alternative 6, for instance, was dropped and replaced by an alternative viewed to better meet the purposes, missions, and related legal responsibilities of the National Park Service and US Fish and Wildlife Service (DeLong 2004). Therefore, I will draw on analyses presented in the BEMP and my own interpretations of the relative merit and associated impacts of alternatives 4 and 6.

Winter feeding is not required or mentioned in any of the NER's establishing legislation or executive orders (USDI 2005). Feeding was instituted to compensate for usurpation of winter range by humans and their livestock, to mitigate subsequent conflicts, and because migration corridors out of the valley were fenced and ranched and those migratory elk eliminated (Allred 1950, Smith et al. 2004). The original size of the NER was inadequate to accommodate the thousands of elk that gathered in Jackson Hole early in the 20th Century (Craighead 1952, Smith et al. 2004). The feeding program modified their behavior by habituating elk to feedgrounds and adjacent environs, creating less uniform distribution of elk across the landscape, and altering migratory patterns (Craighead 1952, Smith 2001). It could be argued that limiting elk movements and migrations may reduce the chance of disease transmission among adjacent wildlife herds and livestock. Yet, thousands of elk winter off feedgrounds in the Jackson herd unit (Lubow and Smith 2004, USDI 2005) and some elk that use feedgrounds currently interchange with adjacent herds.

Table 2. Summary of key elements of alternatives 4 and 6 compared to present conditions or the no action alternative, from USDI 2005*b*.

Element	Current conditions	Alternative 4	Alternative 6
Elk on the NER	Maximum 7,500	4,000-5,000: phased in	Maximum 2,400-3,200
Elk on GTNP	1/3 NER numbers	1,300-1,600	No target, \approx 1,200-1,600
Bison on NER and GTNP	800-1,000+, uncontrolled size	450-500	Average 400
Elk Hunt on NER and GTNP	Yes	Yes	Yes
Bison Hunt NER	No	Yes	Yes
Winter Feeding on NER	Feed 9 of 10 yrs; 70 days/yr	Feed 5 of 10 yrs; phased in over 15 years	Transition to native range (no feeding) in 5 yrs.
Migration	None	Limited increased winter distribution	Natural migration could occur
Use Brucellosis Vaccines on NER	Temporary S19 elk vaccination would end	WGFD use S19 on elk; bison when effective vaccine found	Vaccines not used until found effective (oral for elk)
Transition to Winter Range to Address Disease Prevalence/Risks	No action	Transition in years when feeding does not occur	Transition completely to winter range use within 5 years

I agree with the rankings in the BEMP that Alternative 6 goes much farther than does the plan's preferred Alternative 4 toward achieving the disease management goal (USDI 2005:82, 257), which should include limiting the influence of documented and potential disease incursions on wildlife. Alternative 6 calls for populations of elk and bison managed nearer estimated habitat carrying capacities, phasing out winter feeding, and vaccinations only after improved technology warrants their use against brucellosis. A change in philosophy to managing elk and bison populations within habitat carrying capacity and reliance on natural habitat and forage will not guarantee that brucellosis will be eliminated or diminished to insignificant levels in the Jackson herds. However, it appears certain that the decades-long practices of high population densities, supported by annual feeding in winter, have only served to perpetuate brucellosis and led to transmission of *B. abortus* to cattle herds and loss of Wyoming's brucellosis free status. Consequently, the USDA Animal and Plant Health Inspection Service submitted comments to USDI, during the interagency review period in 2005, in support of

Alternative 6. That letter states, “Alternative 6 provides more risk mitigation and management options which will lower the risk of major adverse impacts that brucellosis and non-endemic, infectious diseases will continue to cause for the elk, bison, and/or livestock populations.” The letter continues, “Moreover, the implementation of Alternative 6 would result in a lower prevalence of brucellosis in the long term as compared to the other alternatives presented.”

The negative effects of winter feeding (Smith 2001, Dunkley and Cattet 2003, Van Deelen 2003), especially exacerbating disease prevalence and susceptibility, would seem to run counter to the disease management goal of the BEMP (USDI 205:34), and sustaining healthy populations of wildlife (USDI 2005:33). Alternative 4 falls short in terms of remedying excessive elk and bison densities in winter, which are at the root of the disease shedding-transmission-infection cycle. Alternative 4 would continue to reinforce elk and bison to crowd onto the NER each fall in anticipation of hay handouts. To break this pattern, periodic reinforcement is not perpetuated under Alternative 6. The BEMP acknowledges the disease liabilities associated with the preferred alternative.

Alternative 4 also calls for continuation of Strain 19 vaccination of elk. Vaccination programs designed to eliminate or greatly reduce brucellosis in feedground elk have arguably failed to reduce disease prevalence, certainly to levels of 0-3% found in elk that do not use winter feedgrounds. Ballistic or hand vaccination requires that elk be baited or fed for ready access. Roffe et al. (2004) succinctly put into context the danger of therapeutic approaches to wildlife disease management, “Even if long-term vaccination was part of a successful program to eventually eliminate brucellosis, this type of management could contribute to maintenance or spread of other diseases. Persisting with this management paradigm could severely hamper our ability to respond to new wildlife disease incursions, especially those for which effective vaccines are nonexistent.”

As I noted in my review of winter feeding of elk in western North America (Smith 2001), “Winter feeding of elk can be viewed as a means of conflict resolution, generally spawned by intense public pressure. It is not based on scientific principle and sustainable resource management policy. Administrators may see winter feeding as the least painful remedy for producing immediate results to appease differing groups: agricultural interests that desire rapid resolution to crop damage, and pro-wildlife constituencies that oppose reductions in elk populations despite wildlife-human conflict or dwindling habitat.” Confined to those arenas, winter feeding of elk arguably has been a success at the NER and elsewhere. However, enter infectious, transmissible disease and the winter feeding solution creates more problems than it solves. It is this issue, diseases of ecological and socioeconomic consequence, which will unavoidably navigate decision-makers toward a new management philosophy in Jackson Hole. It will either happen proactively, or regrettably after the incursion of CWD, bovine TB, or some other intolerable disease.

LITERATURE CITED

- Allred, W. J. 1950. Re-establishment of seasonal elk migration through transplanting. Transactions of the North American Wildlife Conference 15:597–611.

- Aune, K., K. Alt, and T. Lemke. 2002. Managing wildlife habitat to control brucellosis in the Montana portion of the GYA. Pages 109-119 in T. J. Kreeger, editor. Brucellosis in elk and bison in the Greater Yellowstone Area. Wyoming Game and Fish Department, Greater Yellowstone Interagency Brucellosis Committee, Cheyenne, Wyoming, USA.
- Barmore, W. J. Jr. 2003. Ecology of ungulates and their winter range in northern Grand Teton National Park: Research synthesis, 1962-1970. National Park Service, Mammoth Hot Springs, Wyoming, USA.
- Berger, J., and S. L. Cain. 1999. Reproductive synchrony in brucellosis-exposed bison in the southern Greater Yellowstone Ecosystem and in noninfected populations. *Conservation Biology* 13:357-366.
- Boyce, M. S. 1989. The Jackson elk herd: intensive wildlife management in North America. Cambridge University Press, Cambridge, United Kingdom.
- Brown, C. 1985. Sand Creek Elk: Population status, movements, and distribution. Idaho Department of Fish and Game, Job Completion Report, Boise, Idaho, USA.
- Cain, S. L., T. J. Roffe, J. Berger, and C. Cunningham. 2001. Reproduction and demography of brucellosis infected bison in the southern Greater Yellowstone Ecosystem. 2000 annual progress report, Grand Teton National Park, Moose, Wyoming, USA.
- Cheville, N.F., D.R. McCullough, and L.R. Paulson. 1998. Brucellosis in the Greater Yellowstone Area. National Research Council, National Academy Press, Washington, D.C., USA.
- Chronic Wasting Disease Task Force. 2002. Plan for assisting states, federal agencies, and tribes in managing chronic wasting disease in wild and captive cervids. USDA, Animal and Plant Health Inspection Service, Washington, D.C., USA.
- Clause, D., S. Kilpatrick, R. Dean, and B. Smith. 2002. Brucellosis-feedground-habitat program: an integrated management approach to brucellosis in elk in Wyoming. Pages 80-96 in T. J. Kreeger, editor. Brucellosis in elk and bison in the Greater Yellowstone Area. Proceedings of the Greater Yellowstone Interagency Brucellosis Committee Symposium. Wyoming Game and Fish Department, Cheyenne, Wyoming, USA.
- Craighead, J. J. 1952. A biological and economic appraisal of the Jackson Hole elk herd. New York Zoological Society and Conservation Foundation, New York, New York, USA.
- Craighead, J. J., G. Atwell, and B. W. O'Gara. 1972. Elk migrations in and near Yellowstone National Park. *Wildlife Monographs* 29.
- Davis, D. S., J. W. Templeton, T. A. Ficht, J. D. Huber, R. D. Angus, and L. G. Adams. 1991. *Brucella abortus* in bison. II. Evaluation of Strain 19 vaccination of pregnant cows. *Journal of Wildlife Diseases* 27:258-264.
- Davis, D. S. 1993. Summary of bison brucellosis research conducted at Texas A&M University 1985-1993. Pages 346-361 in R. E. Walker, editor. Proceedings of the North American Bison Symposium. National Bison Association, Denver, Colorado, USA.
- Davis D., S., J. W. Templeton, T. A. Ficht, J. D. Williams, J. D. Kopec, and L. G. Adams. 1995. Response to the letter to the editor ... Response to the critique of brucellosis in captive bison. *Journal of Wildlife Diseases* 31:111-114.

- Dean, R., M. Goeke, B. Holz, S. Kilpatrick, T. Kreeger, B. Scurlock, S. Smith, E. T. Thorne, and S. Werbelow. 2004. Elk feedgrounds in Wyoming. Unpublished report. Wyoming Game and Fish Department, Cheyenne, Wyoming, USA.
- DeLong, D. 2004. Factors considered in developing alternative management plans, and their relative importance *in the* Bison and Elk Management Planning Document for the National Elk Refuge and Grand Teton National Park. Unpublished report. National Elk Refuge, Jackson, Wyoming, USA.
- Demarais, S., and P. R. Krausman, editors. 2000. Ecology and management of large mammals in North America. Prentice Hall, Upper Saddle River, New Jersey, USA.
- Dunkley, L. and M. R. L. Cattet. 2003. A comprehensive review of the ecological and human social effects of artificial feeding and baiting of wildlife. Canadian Cooperative Wildlife Health Center, Saskatoon, Saskatchewan, Canada.
- Greater Yellowstone Interagency Brucellosis Committee. 1997. White paper. Available at <http://www.nps.gov/gyibc/whitepap.htm>.
- Gross, J. E., and M. W. Miller. 2001. Chronic wasting disease in mule deer: disease dynamics and control. *Journal of Wildlife Management* 65:205-215.
- Gross, J. E., M. W. Miller, and T. J. Kreeger. 1998. Simulating dynamics of brucellosis in elk and bison. Final report, U.S. Geological Survey, Biological Resources Division, Laramie, Wyoming, USA.
- Hamlin, K. L., and M. S. Ross. 2002. Effects of hunting regulation changes on elk and hunters in the Gravelly-Snowcrest Mountains, Montana. Federal Aid Project W-120-R. Montana Fish, Wildlife and Parks, Helena, Montana, USA.
- Hobbs, N. T., G. Wockner, F. J. Singer, G. Wang, L. Zeigenfuss, M. Coughenour, and S. Delgrosso. 2003. Assessing management alternatives for ungulates in the Greater Teton Ecosystem using simulation modeling: final report. Natural Resources Ecology Lab, Colorado State University, Fort Collins, Colorado, USA.
- Honess, R. F., and K. B. Winter. 1956. Diseases of wildlife in Wyoming. Wyoming Game and Fish Department Bulletin 9, Cheyenne, Wyoming, USA.
- Keiter, R. B., and P. H. Froelicher. 1993. Bison, brucellosis, and law in the Greater Yellowstone Ecosystem. *Land and Water Law Review* 28:1-75.
- Kreeger, T. J., W. E. Cook, W. H. Edwards, P. H. Elzer, and S. C. Olsen. 2002. *Brucella abortus* strain RB51 vaccination in elk II. Failure of high dosage to prevent abortion. *Journal of Wildlife Diseases* 38:27-31.
- Lange, R. E. 1982. Psoroptic scabies. Pages 244-247 in E. T. Thorne, N. Kingston, W. R. Jolley, and R. C. Bergstrom, editors. Diseases of wildlife in Wyoming, 2nd edition. Wyoming Game and Fish Department, Cheyenne, Wyoming, USA.
- Lubow, B. C., and B.L. Smith. 2004. Population dynamics of the Jackson elk herd. *Journal of Wildlife Management* 68(4): 810-829.
- Miller, M. W. 2001. Pasteurellosis. Pages 330-339 in E. S. Williams and I. K. Barker, editors. Infectious diseases of wild mammals. Iowa State University Press, Ames, Iowa, USA.
- Miller, M. W. and M. M. Conner. 2005. Epidemiology of chronic wasting disease in free-ranging mule deer: spatial, temporal, and demographic influences on observed prevalence patterns. *J. Wildlife Diseases* 41(2):275-290.

- Miller, M. W., E. S. Williams, N. T. Hobbs, and L. L. Wolfe. 2004. Environmental sources of prion transmission in mule deer. *Emerging Infectious Diseases* 10(6):4-10.
- Miller, M. W., E. S. Williams, C. W. McCarty, T. R. Spraker, T. J. Kreeger, C. T. Larsen, and E. T. Thorne. 2000. Epizootiology of chronic wasting disease in free-ranging cervids in Colorado and Wyoming. *Journal of Wildlife Diseases* 36:676-690.
- Mohler, J. R., and A. Eichhorn. 1913. Immunization against hemorrhagic septicemia. *American Veterinary Review* 42:409-418.
- Murie, O. J. 1951. *The elk of North America*. Stackpole Books, Harrisburg, Pennsylvania, USA.
- Oldemeyer, J. L., R. L. Robbins, and B. L. Smith. 1993. Effect of feeding level on elk weights and reproductive success at the National Elk Refuge. Pages 64-68 *in* Callas, R. L., D. B. Koch, and E. R. Loft, editors. *Proceedings of the Western States and Provinces Elk Workshop*, California Fish and Game Department, Eureka, California, USA.
- Peters, J., J. M. Miller, A. L. Jenny, T. L. Peterson, and K. P. Carmichael. 2000. Immunohistochemical diagnosis of chronic wasting disease in preclinically affected elk from a captive herd. *Journal of Veterinary Diagnostic Investigation* 12:579-582.
- Peterson, M. J. 2003. Infectious agents of concern for the Jackson Hole elk and bison herds: an ecological perspective. Unpublished report. Department of Wildlife and Fisheries Sciences, Texas A&M University, College Station, Texas, USA.
- Peterson, M. J., W. E. Grant, and D. S. Davis. 1991. Bison-brucellosis management: simulation of alternative management strategies. *Journal of Wildlife Management* 55:205-213.
- Queen, C. J. and T. J. Ryder. 1996. Wiggins Fork elk movements study. Unpublished report. Wyoming Game and Fish Department, Cheyenne, Wyoming, USA.
- Ragan, V. E. 2002. The brucellosis eradication program in the United States. Pages 7-15 *in* T. J. Kreeger, editor. *Brucellosis in elk and bison in the Greater Yellowstone Area*. Wyoming Game and Fish Department, Greater Yellowstone Interagency Brucellosis Committee, Cheyenne, Wyoming, USA.
- Roffe, T. J., and B. L. Smith. 1992. Tuberculosis: will it infect our elk? *Bugle* 9(3):86-92.
- Roffe, T. J., L. C. Jones, K. Coffin, M. L. Drew, S. J. Sweeney, S. D. Hagius, P. H. Elzer, and D. Davis. 2004. Efficacy of single calfhood vaccination of elk with *Brucella abortus* Strain 19. *Journal of Wildlife Management* 68:830-836.
- Rudd, W. J., A. L. Ward, and L. L. Irwin. 1983. Do split hunting seasons influence elk migrations from Yellowstone National Park? *Wildlife Society Bulletin* 11: 328-331.
- Rush, W. M. 1932. Bang's disease in Grand Teton National Park buffalo and elk herds. *Journal of Mammalogy* 13:371-372.
- Samuel, M. D., D. O. Joly, M. A. Wild, S. D. Wright, D. L. Otis, R. W. Werge, and M. W. Miller. 2003. Surveillance strategies for detecting chronic wasting disease in free-ranging deer and elk. US Geological Survey, National Wildlife Health Center, Madison, Wisconsin, USA.

- Sawyer, H., and F. Lindzey. 2001. The Sublette mule deer study. Unpublished report. Wyoming Cooperative Wildlife Research Unit, University of Wyoming, Laramie, Wyoming, USA.
- Smith, B.L. 1994a. Out-of-season births of elk calves in Wyoming. *Prairie Naturalist* 26(2):131-136.
- Smith, B. L. 1994b. Population regulation of the Jackson elk herd. PhD dissertation. University of Wyoming, Laramie, USA.
- Smith, B. L. 1998. Antler size and winter mortality of elk: effects of environment, birth year, and parasites. *Journal of Mammalogy* 79:1038-1044.
- Smith, B. L. 2001. Winter feeding of elk in western North America. *Journal of Wildlife Management* 65:173-190.
- Smith, B. L., and S. H. Anderson. 1998. Juvenile survival and population regulation of the Jackson elk herd. *Journal of Wildlife Management* 62:1036-1045.
- Smith, B. L., and S. H. Anderson. 2001. Does dispersal help regulate the Jackson elk herd? *Wildlife Society Bulletin* 29:331-341.
- Smith, B.L., E.C. Cole, and D. S. Dobkin. 2004. Imperfect pasture: A century of change at the National Elk Refuge in Jackson Hole, Wyoming. Grand Teton Natural History Association, Moose, Wyoming, USA.
- Smith, B.L. and R.L. Robbins. 1994. Migrations and management of the Jackson elk herd. National Biological Survey Resource Publication No. 199. USDI, Washington, D.C., USA.
- Smith, B.L., and T. Roffe. 1994. Diseases among elk of the Yellowstone Ecosystem, U.S.A. Pages 162-166 in W. Van Hoven, J. Ebedes, and A. Conroy, editors. *Proceedings of the Third International Wildlife Ranching Symposium*. Center for Wildlife Management, University of Pretoria Press, Pretoria, South Africa.
- Smith, B.L., and T. Roffe. 1997. Evaluation of studies of Strain 19 *Brucella abortus* vaccine in elk: clinical trials and field applications. Final report. National Elk Refuge, US Fish and Wildlife Service, Jackson, Wyoming, USA.
- Smith, B. L., E. K. Cole, and D. S. Dobkin. 2004. Imperfect pasture: A century of change at the National Elk Refuge in Jackson Hole, Wyoming. Grand Teton Natural History Association, Moose, Wyoming, USA.
- Thorne, E. T. 1982. Brucellosis. Pages 54-63 in E. T. Thorne, N. Kingston, W. R. Jolley, and R. C. Bergstrom, editors. *Diseases of wildlife in Wyoming*. Second edition. Wyoming Game and Fish Department, Cheyenne, Wyoming, USA.
- Thorne, E. T. 2001. Brucellosis. Pages 372-396 in E. S. Williams and I. K. Barker, editors. *Infectious disease of wild mammals*. Iowa State University Press, Ames, Iowa, USA.
- Thorne, E. T., T. J. Kreeger. 2002. Management options for the resolution of brucellosis in the GYA. Pages 19-23 in T. J. Kreeger, editor. *Brucellosis in elk and bison in the Greater Yellowstone Area*. Wyoming Game and Fish Department, Greater Yellowstone Interagency Brucellosis Committee, Cheyenne, Wyoming, USA.
- Thorne, E. T., S. G. Smith, K. Aune, D. Hunter, and T. J. Roffe. 1997. Brucellosis – the disease in elk. Pages 33-46 in E. T. Thorne, M. S. Boyce, P. Nicloetti, and T. J. Kreeger, editors. *Brucellosis, bison, elk, and cattle in the Greater Yellowstone Area: Defining the problem, exploring solutions*. Wyoming Game and Fish Department, Cheyenne, Wyoming, USA.

- Toweill, D. E., and J. W. Thomas, editors. 2002. North American elk: ecology and management. Smithsonian Institution Press, Washington, D. C., USA.
- URS. 2003. Administrative draft EIS section disease impact analysis. Bison and elk management plan and EIS. Prepared for U.S. Fish and Wildlife Service. Denver, Colorado, USA.
- USDI. 2005a. Draft Bison and Elk Management Plan and Environmental Impact Statement. National Elk Refuge and Grand Teton National Park, Denver, Colorado, USA.
- USDI. 2005b. Bison and Elk Management Plan and EIS, Planning Update Number Severn. National Elk Refuge and Grand Teton National Park, Denver, Colorado, USA.
- Van Deelen, T. R. 2003. Chronic wasting disease and the science in support of the ban on baiting and feeding deer. Unpublished report. Wisconsin Department of Natural Resources, Rhinelander, Wisconsin, USA.
- Western EcoSystems Technology. 2004. Summary of elk feedgrounds operated by the Wyoming Game and Fish Department. Prepared for the Northern Rockies Resource Center, National Wildlife Federation. Unpublished report. Western EcoSystems Technology, Cheyenne, Wyoming. USA.
- Williams, E. S. 2001. Paratuberculosis and other mycobacterial diseases. Pages 361-371 in E. S. Williams and I. K. Barker, editors. Infectious diseases of wild mammals. Iowa State University Press, Ames, Iowa, USA.
- Williams, E. S., J. K. Kirkwood, and M. W. Miller. 2001. Chronic Wasting disease. Pages 292-301 in E. S. Williams and I. K. Barker, editors. Infectious diseases of wild mammals. Iowa State University Press, Ames, Iowa, USA.
- Williams, E. S., M. W. Miller, T. J. Kreeger, R. H. Kahn, and E. T. Thorne. 2002. Chronic wasting disease of deer and elk: a review with recommendations for management. *Journal of Wildlife Management* 66:551-563.
- Williams, E. S., and S. Young. 1980. Chronic wasting disease of captive mule deer: spongiform encephalopathy. *Journal of Wildlife Diseases* 16:89-98.
- Wilson, M. A., R. M. Duncan, T. J. Roffe, G. E. Nordholm, and B. M. Verlowski. 1995. Pasteurellosis in elk (*Cervus elaphus*): DNA fingerprinting of isolates. *Veterinary Record* 137:195-196.
- Wobeser, G.A. 1994. Investigation and management of disease in wild animals. Plenum Press, New York, USA.
- Wolfe, L. L., M. M. Conner, T. H. Baker, V. J. Dreitz, K. P. Burnham, E. S. Williams, N. T. Hobbs, and M. W. Miller. 2002. Evaluation of antemortem sampling to estimate chronic wasting disease prevalence in free-ranging mule deer. *Journal of Wildlife Management* 66(3):564-573.
- Wyoming Game and Fish Department. 2004. 2003 Annual report of big and trophy game harvest. Cheyenne, Wyoming, USA.